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Thursday, October 21, 2004

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Search Notes

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=> d his

     (FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

     FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004
                E LONGLEY B/AU
L1             33 E4-6
                E LONGLEY J/AU
L2             12 E3,E9
L3            418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
                E SKIN DISEASE/CT
                E E4+ALL
                E E2
                E E3+ALL
L4          70942 "SKIN, DISEASE"+OLD,NT/CT
L5             11 L4 AND L1-2
L6             24 L4 AND L3
                E STEM CELL/CT
                E E3+ALL
L7          30857 STEM CELL+NT/CT
                E CELL/CT
                E E3+ALL
L8          11056 CELL+OLD,NT/CT (L) STEM
L9              6 L5-6 AND L7-8

     FILE 'STNGUIDE' ENTERED AT 14:06:51 ON 21 OCT 2004

     FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004
L10         48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC
                E STEM CELL#/BI
                E STEM CELL/BI
                E STEM CELL/ABEX
L11          4846 (STEM (1A) CELL?)/BIX
L12           710 L10 AND L11
                E LONGLEY B/AU
L13             3 E4
L14             1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
L15             1 L12 AND L13
L16           810 UYCO/PACO
L17             4 L12 AND (L14 OR L16)
L18             4 L15 OR L17

=> b hcap
FILE 'HCAPLUS' ENTERED AT 14:22:30 ON 21 OCT 2004
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FILE COVERS 1907 - 21 Oct 2004  VOL 141 ISS 17
FILE LAST UPDATED: 20 Oct 2004  (20041020/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


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L9   ANSWER 1 OF 6  HCAPLUS  COPYRIGHT 2004 ACS on STN
AN   2002:674642  HCAPLUS
DN   137:210939
ED   Entered STN:  06 Sep 2002
TI   Methods of use of compounds which inhibit the stem cell factor signaling
     pathway
IN   Longley, B. Jack
PA   USA
```

SO U.S. Pat. Appl. Publ., 22 pp., Cont.-in-part of U.S. Ser. No. 306,143.
CODEN: USXXCO
DT Patent
LA English
IC ICM C12Q001-00
NCL 435004000
CC 1-7 (Pharmacology)
Section cross-reference(s): 2
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002123031	A1	20020905	US 1999-474478	19991229
	US 6576812	B1	20030610	US 1999-306143	19990506
	WO 2000067794	A1	20001116	WO 2000-US12405	20000505

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-306143 A2 19990506
US 1999-474478 A2 19991229

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
US 2002123031	ICM	C12Q001-00
	NCL	435004000
US 2002123031	ECLA	C07K016/28A
US 6576812	ECLA	C07K016/28A

AB The invention provides a method of preventing or treating in a subject contact dermatitis which comprises administering to the subject an amount of a compound capable of inhibiting the stem cell factor signaling pathway effective to prevent or treat contact dermatitis so as to thereby prevent or treat contact dermatitis in the subject. The invention also provides a methods of preventing or treating in a subject hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis and bronchospasm, mastocytosis, tumors which express activated kit, and conception.

ST stem cell factor signaling pathway inhibitor therapeutic; dermatitis hyperpigmentation asthma stem cell factor signaling pathway inhibitor; skin inflammation mastocytosis stem cell factor signaling pathway inhibitor; anaphylaxis bronchospasm tumor stem cell factor signaling pathway inhibitor; contraceptive stem cell factor signaling pathway inhibitor

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgA; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgD; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgE; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgG; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgM; stem cell factor signaling pathway inhibitors for)

IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(SCF-cleaving; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems
(anal; stem cell factor signaling pathway inhibitors for)

IT Ligands
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(binding; stem cell factor signaling pathway inhibitors for)

IT **Dermatitis**
(contact; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fusion products; stem cell factor signaling pathway inhibitors for)
IT Digestive tract, neoplasm
(gastrointestinal stromal tumor, inhibitors; stem cell factor signaling pathway inhibitors for)
IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(humanized; stem cell factor signaling pathway inhibitors for)
IT **Skin, disease**
(**hyperpigmentation**; stem cell factor signaling pathway inhibitors for)
IT Allergy
(hypersensitivity; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(injections, i.m.; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(injections, i.p.; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(injections, i.v.; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(injections, s.c.; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(intestinal; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(intralesional; stem cell factor signaling pathway inhibitors for)
IT Skin
(keratinocyte; stem cell factor signaling pathway inhibitors for)
IT Dimerization
(kit; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(liposomes; stem cell factor signaling pathway inhibitors for)
IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(mucosal; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(nasal; stem cell factor signaling pathway inhibitors for)
IT **Gamete and Germ cell**
(neoplasm, inhibitors; **stem** cell factor signaling pathway inhibitors for)
IT Mast cell
(neoplasm, mastocytoma; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(ophthalmic; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(oral; stem cell factor signaling pathway inhibitors for)
IT Drug delivery systems
(otic; stem cell factor signaling pathway inhibitors for)
IT Bronchi, disease
(spasm; stem cell factor signaling pathway inhibitors for)
IT Allergy inhibitors
Anaphylaxis
Anti-inflammatory agents
Antiasthmatics
Antitumor agents
Asthma
Canis familiaris
Contraceptives
Dermatitis
Felis catus
Human
Mast cell
Melanocyte
Neoplasm
Peptidomimetics
Signal transduction, biological
Transformation, genetic
Urticaria
(stem cell factor signaling pathway inhibitors for)
IT Stem cell factor

Transgene
c-Kit (protein)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins
Nucleic acids
Organic compounds, biological studies
Peptides, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems
(topical; stem cell factor signaling pathway inhibitors for)

IT 9004-06-2, Elastase 97501-92-3, Chymase 138359-29-2, Kit tyrosine kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(stem cell factor signaling pathway inhibitors for)

IT 454746-24-8 454746-25-9 454746-26-0 454746-27-1
RL: PRP (Properties)
(unclaimed nucleotide sequence; methods of use of compds. which inhibit the stem cell factor signaling pathway)

L9 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:539652 HCAPLUS
DN 137:88453
ED Entered STN: 19 Jul 2002
TI Pim kinase-related methods for treatment of an allergic response, asthma, and transplant rejection
IN Rothman, Paul; Chen, Peter
PA The **Trustees** of **Columbia University** In the City of New York, USA
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07D
CC 1-7 (Pharmacology)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002055489	A2	20020718	WO 2001-US50535	20011227
	WO 2002055489	A3	20021003		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2004109868	A1	20040610	US 2004-250380	20040120
PRAI	US 2000-258421P	P	20001227		
	WO 2001-US50535	W	20011227		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2002055489	ICM	C07D

AB The invention provides methods for treating an allergic response, asthma, and the onset of transplant rejection in a subject. The methods involve administering an agent which increases the amount and/or the activity of a Pim kinase. The invention also provides a method for determining whether an agent increases the phosphorylation of a Socs-1 protein by a Pim kinase.

ST Pim kinase modulator allergy asthma treatment; transplant rejection treatment Pim kinase modulator; SOCS1 phosphorylation Pim kinase modulator screening

IT Animal cell line
(293T; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Allergy
Allergy inhibitors
Antiasthmatics
Asthma
Drug screening
Inflammation
Macrophage

Pain
Pruritus
Signal transduction, biological
Swelling, biological
Transplant and Transplantation
Transplant rejection
Urticaria
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Interleukin 4
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Nucleic acids
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(SOCS-1 (suppressor of cytokine signaling-1); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(SOCS-2 (suppressor of cytokine signaling-2); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT6 (signal transducer and activator of transcription 6); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(and small mols.; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(bone marrow; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(bone; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(brain; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Bone
(cell, transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(elongin BC; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(heart; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(intestine; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(kidney; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(liver; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(lung; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(muscle; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation
(pancreas; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(pancreatic islet; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Lymphocyte
(plasma cell, transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Phosphorylation, biological
(protein; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Skin
(redness; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Transplant and Transplantation
(skin; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Thymus gland
(thymocyte; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT Blood cell
Bone
Bone marrow
Brain
Cartilage
Eye
Heart
Intestine
Kidney
Liver
Lung
Muscle
Ovary
Pancreas
Pancreatic islet of Langerhans
Skin
Stem cell
Stomach
(transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)
IT 50812-37-8D, Glutathione-S-transferase, Pim-2 kinase fusion products
144376-45-4, Pim-1 protein kinase 270086-00-5, Pim-3 kinase
420790-04-1, Pim-2 protein kinase 420790-04-1D, Pim-2 protein kinase, GST fusion products
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

L9 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:449525 HCAPLUS
DN 137:32066
ED Entered STN: 14 Jun 2002
TI Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy
IN Chang, Yuan; Moore, Patrick S.
PA The **Trustees** of **Columbia University** in the City of New York, USA
SO PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K039-12
ICS A61K039-245; C12P021-06; C07H021-04
CC 15-3 (Immunochemistry)
Section cross-reference(s): 3, 9, 10, 63
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002045744	A1	20020613	WO 2001-US47217	20011207

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	US 2002137020	A1	20020926	US 2000-733728	20001208
	US 6653465	B2	20031125		
	AU 2002026019	A5	20020618	AU 2002-26019	20011207
PRAI	US 2000-733728	A	20001208		
	WO 2001-US47217	W	20011207		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2002045744	ICM	A61K039-12
	ICS	A61K039-245; C12P021-06; C07H021-04
US 2002137020	ECLA	C07K014/03

AB This invention provides an isolated nucleic acid which encodes a Kaposi's sarcoma-associated herpesvirus latency-associated nuclear antigen 2 polypeptide (LANA2) or a fragment thereof and also provides the LANA2 polypeptide. This invention provides an isolated nucleic acid comprising consecutive nucleotides having the sequence of a promoter of Kaposi's sarcoma-associated herpesvirus latency-associated nuclear antigen 2 transcription. This invention also provides a method of inhibiting p53 mediated apoptosis of a cell and a method of producing an antibody which comprises introducing into a cell a replicable vector of the subject invention.

ST Kaposis sarcoma assocd herpesvirus latency nuclear antigen 2; human herpesvirus 8 LANA2 promoter monoclonal antibody

IT Lymph node, disease
(Castleman's; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal tissue culture
B cell (lymphocyte)
Blood analysis
Blood plasma
Blood serum
Canis familiaris
Cavia porcellus
Cerebrospinal fluid
Coliphage .lambda.
Colorimetric indicators
Cosmids
DNA sequences
Embryo, animal
Escherichia coli
Eubacteria
Eukaryota
Fluorescent substances
Gamete and Germ cell
Hematopoietic precursor cell
Human
Human herpesvirus 8
Immunoassay
Labels
Luminescent substances
Lymph node
Lymphocyte
Mammalia
Mus
Mustela putorius furo
Nucleic acid hybridization
Oryctolagus cuniculus
Ovis aries
Plasmids
Primates
Protein sequences
Rattus
Reptilia
Spleen
Tonsil
Urine analysis
YAC (yeast artificial chromosome)
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT p53 (protein)
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Promoter (genetic element)
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT DNA
Gene, microbial
Nucleic acids
RNA
Radionuclides, biological studies
Toxins
cDNA
mRNA
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Antigens
Proteins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(LANA2; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Drug delivery systems
(carriers; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Bone marrow
(cells; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Test kits
(diagnostic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Immunoassay
(enzyme-linked immunosorbent assay; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Immunoassay
(fluorescence; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Cell
(host; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Immunoassay
(immunoblotting; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Drug delivery systems
(immunoconjugates; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide,

polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis
(immunodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Drug delivery systems
(immunotoxins; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT **Skin, disease**
(**lesion**; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Apoptosis
(p53-mediated; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Lymphoma
(primary effusion; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis
(radiodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Genetic vectors
(replicable; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis
(serodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal cell
(somatic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal
(transgenic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Body fluid
(transudates and exudates; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT 437131-44-7P
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT 437131-45-8P 437131-46-9P
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; Kaposi's sarcoma-associated herpesvirus or human

herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT 437132-47-3 437132-48-4 437132-49-5 437132-50-8 437132-51-9 437132-52-0
RL: PRP (Properties)
(unclaimed sequence; kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Ballestas; Science 1999, V284, P641 HCAPLUS
(2) Dittmer; Journal of Virology 1998, V72(10), P8309 HCAPLUS
(3) Dupin; PNAS 1999, V96, P4546 HCAPLUS
(4) Friborg; Nature 1999, V402(23), P889
(5) Katano; Virology 2000, V269, P335 HCAPLUS
(6) Neipel; Journal of Virology 1997, V71(6), P4187 HCAPLUS
(7) Russo; PNAS 1996, V93, P14862 HCAPLUS

L9 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:614258 HCAPLUS
DN 131:227652
ED Entered STN: 26 Sep 1999
TI Human monoclonal antibodies from tetroma cells
IN Trakht, Ilya
PA The **Trustees** of **Columbia University** In the City of New York, USA
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM G01N033-53
ICS G01N033-567; C07K016-00; A61K039-395; A61K039-42
CC 15-1 (Immunochemistry)
Section cross-reference(s): 1, 8, 14, 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9947929	A1	19990923	WO 1999-US5828	19990318
	W: AU, CA, JP, MX, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6197582	B1	20010306	US 1998-40833	19980318
	CA 2323681	AA	19990923	CA 1999-2323681	19990318
	AU 9931889	A1	19991011	AU 1999-31889	19990318
	EP 1064551	A1	20010103	EP 1999-913925	19990318
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002507398	T2	20020312	JP 2000-537073	19990318
PRAI	US 1998-40833	A2	19980318		
	WO 1999-US5828	W	19990318		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9947929	ICM	G01N033-53
	ICS	G01N033-567; C07K016-00; A61K039-395; A61K039-42

AB The author discloses the preparation of antibody-non-producing heteromyeloma and trioma cells from the fusion of human and mouse myeloma and human lymphoid cells, resp. The trioma cell fusion partner, when again fused with a human lymphoid cell, provides a tetroma capable of producing a monoclonal antibody having specific binding affinity for antigen. The invention thus provides a method of producing a monoclonal antibody with specificity for cells, tissue, or disease state. The author also discloses therapeutic and diagnostic application of these tetroma-derived monoclonal antibodies.

ST heteromyeloma trioma tetroma monoclonal antibody

IT Hybridoma
(B-cell, heteromyeloma; for fusion with lymphoid cells in production of monoclonal antibodies)

IT Animal cell line
(B6B11; for fusion with lymphoid cells in production of monoclonal antibodies)

IT Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES

(Uses)
(M, monoclonal; to breast and prostate cancer antigens)
IT Animal cell line
(MFP-2; for fusion with lymphoid cells in production of monoclonal antibodies)
IT Transplant rejection
(allotransplant; tetroma-derived monoclonal antibodies as therapy for)
IT Toxins
RL: ADV (Adverse effect, including toxicity); ANT (Analyte); ANST (Analytical study); BIOL (Biological study)
(anthrax; tetroma production of monoclonal antibodies to)
IT Antibodies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(autoantibodies; tetroma-derived monoclonal antibodies as therapy against)
IT Thyroid gland, disease
(autoimmune thyroiditis; tetroma-derived monoclonal antibodies as therapy for)
IT Magnetic materials
(beads, conjugates with tetroma-derived monoclonal antibodies; for disease diagnosis)
IT Diagnosis
(cancer; by tetroma-derived monoclonal antibodies to tumor antigens)
IT Chromophores
Fluorescent dyes
(conjugates with tetroma-derived monoclonal antibodies; for disease diagnosis)
IT Cytotoxic agents
(conjugates with tetroma-derived monoclonal antibodies; therapeutic application of)
IT Enzymes, biological studies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(conjugates, with tetroma-derived monoclonal antibodies; for disease diagnosis)
IT Metabolism, animal
(disorder, enzyme dysfunction; tetroma-derived monoclonal antibodies as therapy for)
IT Hormones, animal, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(dysfunction; tetroma-derived monoclonal antibodies as therapy for)
IT Lymphocyte
(for fusion with heteromyeloma or trioma in production of monoclonal antibodies)
IT Multiple myeloma
(for fusion with lymphoid cells in production of monoclonal antibodies)
IT B cell (lymphocyte)
Erythroblast
Macrophage
Monocyte
T cell (lymphocyte)
(for fusion with trioma in production of monoclonal antibodies)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(for monoclonal antibody heavy and light chains from tetroma)
IT Gel electrophoresis
Gel permeation chromatography
(for separation of tetroma-derived monoclonal antibody from cognate antigen)
IT Transplant and Transplantation
(graft-vs.-host reaction; tetroma-derived monoclonal antibodies as therapy for)
IT Thyroid gland, neoplasm
Thyroid gland, neoplasm
(inhibitors; tetroma-derived monoclonal antibodies as)
IT Drug delivery systems
(liposomes; as carriers of tetroma-derived monoclonal antibodies)
IT Animal cell
(mammalian; tetroma production of monoclonal antibodies to antigens specific to)
IT Antitumor agents
(mammary gland; tetroma-derived monoclonal antibodies as)
IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(monoclonal, labeled, tetroma-derived; for diagnostic and therapeutic applications)

IT Antibodies
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; fusion of human lymphoid cells with heteromyeloma and trioma for production of)
IT Mammary gland
Mammary gland
Prostate gland
Prostate gland
(neoplasm, inhibitors; tetroma-derived monoclonal antibodies as)
IT Immune complexes
RL: ANT (Analyte); ANST (Analytical study)
(of antigens with tetroma-derived monoclonal antibodies)
IT Immunotherapy
(of autoimmune disease with tetroma-derived monoclonal antibodies)
IT Cell fusion
(of heteromyeloma and trioma with human lymphoid cells in production of monoclonal antibodies)
IT Cryopreservation
(of human lymphoid cells for fusion with heteromyeloma and trioma in production of monoclonal antibodies)
IT Antigens
RL: ANT (Analyte); ANST (Analytical study)
(preparation of tetroma and production of monoclonal antibodies to)
IT Antitumor agents
(prostate gland; tetroma-derived monoclonal antibodies as)
IT Shock (circulatory collapse)
(septic; tetroma-derived monoclonal antibodies as therapy for)
IT Venoms
(snake; tetroma-derived monoclonal antibodies as therapy against)
IT Venoms
(spider; tetroma-derived monoclonal antibodies as therapy against)
IT Spleen
(splenocyte; for fusion with heteromyeloma or trioma in production of monoclonal antibodies)
IT **Lupus erythematosus**
(systemic; tetroma-derived monoclonal antibodies as therapy for)
IT Toxins
RL: ADV (Adverse effect, including toxicity); ANT (Analyte); ANST (Analytical study); BIOL (Biological study)
(tetanus; tetroma production of monoclonal antibodies to)
IT Animal virus
Bacteria (Eubacteria)
Escherichia coli
Eukaryote (Eukaryotae)
Fungi
Insect (Insecta)
Klebsiella
(tetroma production of monoclonal antibodies to)
IT Enzymes, analysis
Immunoglobulins
Nucleic acids
RL: ANT (Analyte); ANST (Analytical study)
(tetroma production of monoclonal antibodies to)
IT Animal cell
Animal tissue
Neoplasm
(tetroma production of monoclonal antibodies to antigens specific to)
IT Anti-AIDS agents
Antibacterial agents
Antirheumatic agents
Antiviral agents
Fungicides
(tetroma-derived monoclonal antibodies as)
IT Bacillus anthracis
Cryptococcus (fungus)
Ebola virus
Hantavirus
Herpesviridae
Human T-lymphotropic virus 1
Human T-lymphotropic virus 2
Human papillomavirus
Influenza virus
Staphylococcus
Streptococcus

(tetroma-derived monoclonal antibodies as therapy against)
IT Autoimmune disease
Sepsis
Septicemia
(tetroma-derived monoclonal antibodies as therapy for)
IT CD3 (antigen)
CD4 (antigen)
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(tetroma-derived monoclonal antibodies as therapy for immune dysfunction mediated by)
IT Immunoassay
(tetroma-derived monoclonal antibodies in)
IT Hybridoma
(tetroma; production of monoclonal antibodies by)
IT Antitumor agents
Antitumor agents
(thyroid; tetroma-derived monoclonal antibodies as)
IT Hybridoma
(trioma; for fusion with lymphoid cells in production of monoclonal antibodies)
IT Antigens
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(tumor-associated; preparation of tetroma and production of monoclonal antibodies to)
IT Spider
(venom; tetroma-derived monoclonal antibodies as therapy against)
IT Radionuclides, biological studies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(with tetroma-derived monoclonal antibodies; for disease diagnosis)
IT 107231-12-9, Botulin
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(tetroma-derived monoclonal antibodies as therapy against)
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Larrick; US 5001065 A 1991 HCAPLUS
(2) Teng, N; Proc Natl Acad Sci USA December 1983, V80, P7308 MEDLINE

L9 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:1558 HCAPLUS
DN 128:58320
ED Entered STN: 02 Jan 1998
TI Human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9
IN Lieberman, Howard B.; Hopkins, Kevin M.
PA **Trustees of Columbia University, USA**; Lieberman, Howard B.; Hopkins, Kevin M.
SO PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N
CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 1, 6, 13
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9746661	A2	19971211	WO 1997-US8798	19970509
	W: AU, CA, JP, MX, US				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 5882862	A	19990316	US 1996-644034	19960509
	CA 2261637	AA	19971211	CA 1997-2261637	19970509
	AU 9746431	A1	19980105	AU 1997-46431	19970509
PRAI	US 1996-644034	A2	19960509		
	WO 1997-US8798	W	19970509		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9746661	ICM	C12N
US 5882862	ECLA	C07K014/47A26; C12Q001/68M; C12Q001/68M6B

AB This invention provides isolated nucleic acids which encode a wild-type and a mutant human homolog of RAD9. This invention also provides purified and mutant wild-type human homologs of RAD9. The wild-type human homolog of the fission yeast RAD9 checkpoint control gene was purified from a human placental cDNA library by in situ plaque hybridization. Sequence

anal. showed the full-length clone to have a 1176-bp coding region for a protein of 391 amino acids, which is 26% identical and 52% similar at the amino acid level to S. pombe Rad9. Human RAD9 was localized to chromosomal region 11q13.1-11q13.2. A plasmid vector designated pHRAD9-1 was made by cleaving DNA which encodes a wild-type human RAD9 homolog with HindIII and NotI, creating blunt ends and inserting the DNA into the SmaI site of pART1. This invention also provides methods for determining whether a subject has radiosensitivity; for predicting the effect of radiation therapy or chemotherapy on a subject; for detecting whether a subject has a predisposition to cancer; for treating a subject who is radiosensitive and for preventing or treating cancer in a subject who is radiosensitive. This invention further provides a method of detecting cervical carcinoma in a subject.

ST human gene RAD9 sequence radiosensitivity chemotherapy; cervical cancer detection human gene RAD9; cell cycle gene RAD9 human

IT Gene, animal
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(RAD9; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Nervous system
(ataxia telangiectasia, treatment of; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Diagnosis
(cancer, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Uterus, neoplasm
(cervix, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Amniotic fluid
Blood analysis
Brain, neoplasm
Chemotherapy
Lung, neoplasm
Melanoma
Radiotherapy
Susceptibility (genetic)
(determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Neoplasm
(diagnosis, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Embryo, animal
(fetus, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Muscle
(fiber, gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT cDNA sequences
(for human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Proteins, specific or class
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(gene RAD9; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Bone marrow
Gamete and Germ cell
Intestine
Liver
(gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Chromosome
(human 13, human RAD9 homolog mapping to human chromosome 11q13; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Genetic mapping

(human RAD9 homolog mapping to human chromosome 11q13; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Antitumor agents
Cell cycle
Cosmids
Gene therapy
Genetic vectors
Virus vectors
YAC (yeast artificial chromosome)
(human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Mammary gland
(neoplasm, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Protein sequences
(of human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Plasmid vectors
(pHRAD9-1; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Diagnosis
(prenatal, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Kidney, neoplasm
(renal cell carcinoma, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Gamma ray
UV radiation
(sensitivity; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT **Cell**
(**stem**, gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Mammal (Mammalia)
Mouse
(transgenic, for radiotherapy and chemotherapy sensitivity research; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT **Skin, disease**
(**xeroderma pigmentosum**, treatment of; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 184594-71-6P, Protein (human brain gene HRAD9)
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 127-07-1, Hydroxyurea
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 184595-34-4P, GenBank U53174
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

L9 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1994:646298 HCAPLUS
DN 121:246298
ED Entered STN: 26 Nov 1994
TI Anhydroretinol and derivatives thereof as antagonists of retinol and 14-hydroxyretroretinol
IN Hammerling, Ulrich; Buck, Jochen; Derguini, Fadila; Nakanishi, Koji
PA Sloan-Kettering Institute for Cancer Research, USA; **Trustees of Columbia University in the City of New York**
SO PCT Int. Appl., 73 pp.
CODEN: PIXXD2

DT Patent
LA English
IC ICM A61K031-015
ICS A61K031-045; A61K031-75; A61K031-22; A61K031-23
CC 1-6 (Pharmacology)
Section cross-reference(s): 15
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9420081	A1	19940915	WO 1994-US2450	19940308
	W: AU, CA, FI, HU, JP, KR, NO, NZ, PL, RO, RU				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9463614	A1	19940926	AU 1994-63614	19940308
PRAI	US 1993-27880		19930308		
	WO 1994-US2450		19940308		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9420081	ICM	A61K031-015
	ICS	A61K031-045; A61K031-75; A61K031-22; A61K031-23

AB This invention provides methods of inhibiting the growth of cells, of treating a subject having a disease characterized by an uncontrolled growth of cells and of blocking an immune response as well as related inflammatory responses in a subject which comprises administering anhydroretinol and derivs. Anhydroretinol antagonizes retinol and 14-hydroxyretroretinol. All-trans anhydroretinol was an antagonist and reversible inhibitor of human B lymphoblastoid tumor cell proliferation. Retro retinyl Me ether reversed the growth-promoting effect of retinol in activated mouse T lymphocytes at 10-5-10-8M.

ST anhydroretinol antagonist retinol hydroxyretroretinol; cell proliferation inhibitor anhydroretinol; immune response inhibitor anhydroretinol; antiinflammatory anhydroretinol; neoplasm inhibitor anhydroretinol

IT Allergy inhibitors
Antidiabetics and Hypoglycemics
Cell proliferation
Immunosuppressants
Inflammation inhibitors
Neoplasm inhibitors
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT **Dermatitis**
(inhibition of chemical; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Venoms
(inhibition of contact allergy to insect; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Insect
(inhibition of contact allergy to venom of; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Poison ivy
Poison oak
Poison sumac
(inhibition of contact allergy to; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Monocyte
(inhibition of immune response mediated by; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Autoimmune disease
Kidney, disease
Psoriasis
(treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Lymphocyte
(B-cell, transformed; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Neoplasm inhibitors
(B-cell lymphoma, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune

response and inflammation)

IT Antigens
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(CD4, T-cells pos. for; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Antigens
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(CD8, T-cells pos. for; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(CRBP-I (cellular retinol-binding protein I), anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Lymphocyte
(T-cell, activated; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Inflammation inhibitors
(antirheumatics, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Immunity
(cell-mediated, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Allergy inhibitors
(contact, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Immunity
(disorder, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Transplant and Transplantation
(graft-vs.-host reaction, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Immunity
(humoral, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Neoplasm inhibitors
(leukemia, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Neoplasm inhibitors
(mammary gland, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Neoplasm inhibitors
(myelogenous leukemia, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT **Hematopoietic precursor cell**
(myeloid, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Mammary gland
(neoplasm, inhibitors, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(retinol-binding, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT Thyroid gland, disease
(thyroiditis, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and

immune response and inflammation)
IT Bone marrow
Organ
(transplant, immune response to; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
IT 68-26-8, Retinol 153219-51-3
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
IT 1224-78-8P, Anhydroretinol
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
IT 158730-82-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

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AN 2004-122909 [12] WPIX
DNC C2004-049553
TI Inducing differentiation of embryonic **stem cells**, useful for treating neurodegenerative diseases, comprises contacting the cells with rostralizing and/or caudalizing and dorsalizing or ventralizing embryonic signaling factors.
DC B04 D16
IN JESSEL, T M; LIEBERAM, I; WICHTERLE, H
PA (UYCO) UNIV COLUMBIA NEW YORK
CYC 101
PI WO 2004007665 A2 20040122 (200412)* EN 76 C12N000-00
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT

RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW

ADT WO 2004007665 A2 WO 2003-US20399 20030626
PRAI US 2002-196882 20020716
IC ICM C12N000-00
AB WO2004007665 A UPAB: 20040418
NOVELTY - Inducing differentiation of an embryonic **stem cell** into a differentiated neural cell by contacting the embryonic **stem cell** with amounts of a rostralizing and/or caudalizing embryonic signaling factor and a dorsalizing or ventralizing embryonic signaling factor effective to produce a differentiated neural cell, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) producing differentiated neural cells;
(2) differentiated neural cells or neural progenitor cells produced by the methods cited above;
(3) repopulating a spinal cord in a subject;
(4) treating nervous tissue degeneration in a subject;
(5) a transgenic non-human animal line containing the embryonic **stem cells** cited above, where the cells express enhanced green fluorescent protein (eGFP);
(6) isolating a population of differentiated neural cells; and
(7) identifying an agent for use in treating a condition associated with motor neuron degeneration.
ACTIVITY - Neuroprotective; Vulnerary; Muscular-Gen.; Nootropic; Anticonvulsant; Antiparkinsonian; Cerebroprotective.
No biological data given.
MECHANISM OF ACTION - Cell therapy.
USE - The method is useful in inducing differentiation of embryonic **stem cells**. The differentiated neural cells or the neural progenitor cells are used in analyzing neuron development, function and death, or in monitoring synaptic differentiation (claimed). The cells and the methods may also be used in treating nervous tissue degeneration, such as a peripheral neuropathy or a neurodegenerative disease, or in identifying agents for use in treating a condition associated with neuron degeneration. Diseases that may be treated include amyotrophic lateral sclerosis, neural trauma, paraneoplastic syndrome, polio, postpolio syndrome, progressive bulbar palsy, spinal muscular atrophy, Alzheimer's disease, Binswanger's disease, Huntington's chorea, multiple sclerosis, myasthenia gravis, Parkinson's disease or Pick's disease.
Dwg.0/10
FS CPI
FA AB; DCN
MC CPI: B03-A; B04-F0200E; B10-C04A; B11-C08E1; B12-K04E; B14-G02D; B14-J01A3; B14-J01A4; B14-J05; **B14-N17B**; B14-S01; D05-H08; D05-H09; D05-H14B2

L18 ANSWER 2 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN
AN 2004-098824 [10] WPIX
DNC C2004-040709
TI Inducing differentiation of embryonic **stem cells** into differentiated neural cells, by contacting **stem cells** with rostralizing and/or caudalizing embryonic signaling factor and dorsalizing or ventralizing signaling factor.
DC B04 D16 P25
IN JESSEL, T M; LIEBERAM, ; WICHTERLE, H; JESSELL, T M; LIEBERAM, I
PA (**UYCO**) UNIV COLUMBIA NEW YORK; (JESS-I) JESSELL T M; (LIEB-I) LIEBERAM I; (WICH-I) WICHTERLE H
CYC 102
PI US 2004014210 A1 20040122 (200410)* 34 C12N005-06
WO 2004007665 A2 20040122 (200412) EN 76 C12N000-00
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW
AU 2003247809 A1 20040202 (200453) A47B083-02
ADT US 2004014210 A1 US 2002-196882 20020716; WO 2004007665 A2 WO 2003-US20399 20030626; AU 2003247809 A1 AU 2003-247809 20030626
FDT AU 2003247809 A1 Based on WO 2004007665
PRAI US 2002-196882 20020716
IC ICM A47B083-02; C12N000-00; C12N005-06
ICS A47B003-14; A61K031-203; A61K038-17; C12N005-08
AB US2004014210 A UPAB: 20040901

NOVELTY - Inducing (M1) differentiation of an embryonic **stem cell** into a differentiated neural cell, by contacting the embryonic **stem cell** with a rostralizing and/or caudalizing embryonic signaling factor and a dorsalizing or ventralizing embryonic signaling factor, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a differentiated neural cell (I) produced by (M1);

(2) producing (M2) differentiated neural cells, by obtaining or generating a culture of embryonic **stem cells**, contacting the culture embryonic **stem cells** with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, contacting the neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells, and optionally contacting the differentiated neural cells with at least one neurotrophic factor;

(3) a population of cells (II) comprising differentiated neural cells produced by M2;

(4) repopulating (M3) a spinal cord in a subject, by preparing differentiated neural cells using method M2, and transplanting the cells into the spinal chord of the subject;

(5) treating (M4) nervous tissue degeneration in a subject by transplanting cells prepared according to M2 into the subject;

(6) neural progenitor cells (NP) produced by obtaining or generating a culture of embryonic **stem cells**, contacting the culture of embryonic **stem cells** with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, and optionally contacting the neural progenitor cells with a dorsalizing or ventralizing embryonic signaling factor;

(7) differentiated neural cells (III) produced by obtaining or generating a culture of embryonic **stem cells**, contacting the culture of embryonic **stem cells** with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, contacting the neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells, and optionally contacting the differentiated neural cells with at least one neurotrophic factor;

(8) a transgenic non-human animal line containing (III), where the cells express enhanced green fluorescent protein (eGFP);

(9) isolating a population of differentiated neural cells, involves obtaining or generating a culture of embryonic **stem cells** that express eGFP, contacting the culture of embryonic **stem cells** with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells that express eGFP, contacting neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells that express eGFP, optionally contacting the differentiated neural cells with at least one neurotrophic factor, detecting expression of eGFP in the differentiated neural cells, and isolating the differentiated neural cells that express eGFP; and

(10) identifying an agent for use in treating a condition associated with motor neuron degeneration, by obtaining or generating a culture of embryonic **stem cell**, contacting the culture of embryonic **stem cells** with retinoic acid to produce neuronal progenitor cells, activating a Hedgehog signaling pathway in the neuronal progenitor cells to produce motor neurons, where some or all of the motor neurons are degenerated, contacting the degenerated motor neurons with the candidate agent and determining if the agent enhances regeneration of some or all of the degenerated motor neurons.

ACTIVITY - CNS-Gen.; Neuroprotective; Cerebroprotective; Vulnerary; Nootropic; Anticonvulsant; Antiparkinsonian; Muscular-Gen.

No biological data given.

MECHANISM OF ACTION - Cell therapy.

USE - M1 is useful for inducing differentiation of an embryonic **stem cell** into a differentiated neural cell. The embryonic **stem cell** is murine embryonic **stem cell** or human embryonic **stem cell.** The differentiated neural cell is transplanted into the spinal cord of the subject. The subject is an embryo or human. The subject has nervous tissue degeneration. The nervous tissue degeneration is a peripheral neuropathy or a neurodegenerative disease. The peripheral neuropathy is associated with a condition chosen from amyotrophic lateral sclerosis (ALS), neural trauma, paraneoplastic syndrome, polio, postpolio syndrome, progressive bulbar palsy, and spinal muscular atrophy (SMA), preferably ALS or SMA. The neurodegenerative disease is chosen from Alzheimer's disease, amyotrophic lateral sclerosis (Lou Gehrig's disease), Binswanger's

disease, Huntington's chorea, multiple sclerosis, myasthenia gravis, Parkinson's disease, and Pick's disease. NP and (III) is useful or analyzing neuron development, function and death, or in monitoring synaptic differentiation (all claimed).
Dwg.0/10

FS CPI GMPI
FA AB; DCN
MC CPI: B03-A; B04-F02; B04-F0200E; B11-C07B3; B11-C08E1; B12-K04A5; B12-K04E; B14-G02D; B14-J01A3; B14-J01A4; B14-J05; B14-N16; **B14-N17B**; B14-S01; D05-H08; D05-H09; D05-H14B2

L18 ANSWER 3 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN
AN 2000-687622 [67] WPIX
DNC C2000-209364
TI Prevention and treatment of contact dermatitis, hyperpigmentation, cutaneous inflammation and other conditions, comprises inhibiting the **stem cell** factor signaling pathway.
DC B04 D16 D21
IN **LONGLEY, B J**
PA (**UYCO**) UNIV COLUMBIA NEW YORK; (LONG-I) LONGLEY B J
CYC 90
PI WO 2000067794 A1 20001116 (200067)* EN 72 A61K039-395
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000048253 A 20001121 (200112) A61K039-395
US 2002123031 A1 20020905 (200260) C12Q001-00
US 6576812 B1 20030610 (200340) G01N033-00
ADT WO 2000067794 A1 WO 2000-US12405 20000505; AU 2000048253 A AU 2000-48253 20000505; US 2002123031 A1 CIP of US 1999-306143 19990506, US 1999-474478 19991229; US 6576812 B1 US 1999-306143 19990506
FDT AU 2000048253 A Based on WO 2000067794
PRAI US 1999-474478 19991229; US 1999-306143 19990506
IC ICM A61K039-395; C12Q001-00; G01N033-00
ICS A01K067-00; A01K067-027; A01K067-033; C07K016-00; C12Q001-70
AB WO 200067794 A UPAB: 20001223
NOVELTY - Preventing or treating diseases comprises administering a compound capable of inhibiting the **stem cell** factor signaling pathway.

DETAILED DESCRIPTION - Preventing or treating contact dermatitis, hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis or bronchospasm, mastocytosis, urticaria, hypersensitivity, airway inflammation, interstitial cystitis or a tumor which expresses activated kit comprises administering to the subject a compound capable of inhibiting the **stem cell** factor signaling pathway effective to prevent or treat the conditions.

INDEPENDENT CLAIMS are also included for the following:

(1) providing contraception comprising administering a compound capable of inhibiting the cell factor signaling pathway effective to prevent conception;

(2) desensitizing a subject to an agent comprising administering to the subject, during the afferent phase of an immune response, a compound capable of inhibiting the **stem cell** factor signaling pathway effective to desensitize the subject;

(3) identifying a composition, compound or procedure which can produce a skin response comprising administering the compound or composition or applying the procedure to transgenic mice which express endogenous epidermal **stem cell** factor and analyzing the skin of the transgenic mice for a response;

(4) identifying a composition, compound or procedure which can reduce skin response in a subject comprises administering the composition or compound or applying the procedure to the transgenic mice which express endogenous epidermal **stem cell** factor and which had been induced to produce a skin disease and analyzing the skin to determine the reduction of the skin response;

(5) identifying a compound, composition or procedure which can reduce radiation damage to skin comprises administering the composition or compound or applying the procedure to the skin of the transgenic mice which express endogenous epidermal **stem cell** factor, subjecting the skin of the transgenic mice and control mice to radiation and analyzing the effects of the composition, compound or procedure on reducing skin radiation damage;

(6) a composition for treating human skin diseases comprising a

compound that can treat skin diseases of the transgenic mice which express endogenous epidermal **stem cell** factor and a carrier, wherein the compound specifically targets the epidermal **stem cell** factor or its receptor.

ACTIVITY - Dermatological; antiinflammatory; antiasthmatic; antiinflammatory; antiallergic; immunosuppressive; cytostatic. No biological data is given.

MECHANISM OF ACTION - **Stem cell** factor signaling pathway inhibitor.

USE - The methods can be used to treat and/or prevent contact dermatitis, hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis or bronchospasm , mastocytosis, urticaria, hypersensitivity, airway inflammation e.g. rhinitis, interstitial cystitis, a tumor which expresses activated kit wherein the tumor is e.g. a gastrointestinal stromal tumor or a germ cell tumor or radiation damage. The method may also be used to provide contraception (claimed).

Dwg.0/13

FS CPI

FA AB; DCN

MC CPI: B04-E01; B04-G01; B04-G21; B04-H16; B04-M01; B04-N04; B11-C08E2; B12-K04E; B14-C03; B14-H01; B14-K01; B14-K01A; B14-N07B; **B14-N17**; **B14-N17C**; B14-P01; B14-S06; D05-H09; D05-H11A; D05-H12; D05-H16A

L18 ANSWER 4 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN

AN 1999-277439 [23] WPIX

DNC C1999-081536

TI New peptides based on an advanced glyation end product receptor are useful for treating Alzheimer's disease and Down's syndrome.

DC B04 D16

IN LAMSTER, I; SCHMIDT, A M; STERN, D; YAN, S D

PA (**UYCO**) UNIV COLUMBIA NEW YORK; (LAMS-I) LAMSTER I; (SCHM-I) SCHMIDT A M; (STER-I) STERN D; (YANS-I) YAN S D

CYC 24

PI WO 9918987 A1 19990422 (199923)* EN 99 A61K038-00
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 9897958 A 19990503 (199937)
EP 1023080 A1 20000802 (200038) EN A61K038-00
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2001519401 W 20011023 (200202) 84 C07K007-04
US 2001053357 A1 20011220 (200206) A61K038-04
US 6555651 B2 20030429 (200331) A61K038-00

ADT WO 9918987 A1 WO 1998-US21346 19981009; AU 9897958 A AU 1998-97958 19981009; EP 1023080 A1 EP 1998-952204 19981009, WO 1998-US21346 19981009; JP 2001519401 W WO 1998-US21346 19981009, JP 2000-515619 19981009; US 2001053357 A1 US 1997-948131 19971009; US 6555651 B2 US 1997-948131 19971009

FDT AU 9897958 A Based on WO 9918987; EP 1023080 A1 Based on WO 9918987; JP 2001519401 W Based on WO 9918987

PRAI US 1997-948131 19971009

IC ICM A61K038-00; A61K038-04; C07K007-04
ICS A61K009-06; A61K009-08; A61K009-12; A61K009-14; A61K039-395; A61K047-48; A61P001-02; A61P003-10; A61P009-10; A61P015-10; A61P017-02; A61P021-02; A61P025-28; A61P029-00; A61P035-00; A61P037-06; A61P043-00; C07K005-00; C07K014-00; C07K014-705; C07K016-00; C07K017-00; C12P021-08

AB WO 9918987 A UPAB: 19990616

NOVELTY - Novel isolated peptides (I) having an amino acid sequence corresponding to an amino acid sequence of a V-domain of a receptor for an advanced glycation end product (AGEP), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a composition comprising (I) linked to an antibody or its portion;

(2) a method for inhibiting an amyloid-beta peptide (ABP) interaction with a receptor for AGEP when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the ABP with the receptor for AGEP;

(3) a method for inhibiting degeneration of a neuronal cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit degeneration of the neuronal cell;

(4) a method for inhibiting formation of an ABP fibril on a cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and

thereby inhibit formation of the ABP fibril on a cell;

(5) a method for inhibiting extracellular assembly of an ABP into a fibril which comprises contacting the ABP with an inhibitor of the interaction of an ABP with another ABP so as to inhibit the interaction and thereby inhibit extracellular assembly of an ABP into a fibril;

(6) a method for inhibiting aggregation of ABP on the surface of a cell which comprises contacting the ABP with an inhibitor of the interaction of the ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit aggregation of ABP on the surface of a cell;

(7) a method for inhibiting infiltration of a microglial cell into senile plaques which comprises contacting the microglial cell with an inhibitor of the interaction of an ABP with a receptor for AGEP on the surface of the microglial cell, so as to inhibit the interaction and thereby inhibit infiltration of a microglial cell into senile plaques;

(8) a method for inhibiting activation of a microglial cell by an ABP which comprises contacting the microglial cell with an inhibitor of the interaction of the ABP with a receptor for AGEP on the surface of the microglial cell so as to inhibit the interaction and thereby inhibit activation of a microglial cell;

(9) a method for treating a subject with a condition associated with an interaction of an ABP with a receptor for AGEP on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the ABP with the receptor for AGEP;

(10) a method for evaluating the ability of an agent to inhibit binding of an ABP with a V-domain of a receptor for AGEP on the surface of a cell which comprises:

(a) contacting the cell with the agent and ABP;

(b) determining the amount of ABP bound to the cell; and

(c) comparing the amount of bound ABP determined in (b) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit the binding of ABP to the V-domain of the receptor for AGEP on the surface of the cell;

(11) a method for inhibiting activation of a NF-kappaB gene in a cell which comprises contacting the cell with an inhibitor of the interaction of ABP with a receptor for AGEP on the cell so as to inhibit the interaction and thus inhibit activation of NF-kappaB in the cell;

(12) a method for inhibiting periodontal disease in a subject which comprises administering topically to the subject a pharmaceutical composition which comprises soluble receptor for an AGEP (sRAGE) to accelerate wound healing and thereby inhibit periodontal disease;

(13) a method for inhibiting an AGEP's interaction with a receptor for AGEP when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the AGEP with the receptor for AGEP;

(14) a method for treating a subject with a condition associated with an interaction of an AGEP with a receptor for AGEP on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the AGEP with the receptor for AGEP.

USE - The methods can be used for treating conditions associated with an interaction of an ABP or an AGEP with a receptor for AGEP, e.g. diabetes, Alzheimer's disease, senility, renal failure, hyperlipidemic atherosclerosis, neuronal cytotoxicity, Down's syndrome, dementia associated with head trauma, amyotrophic lateral sclerosis, multiple sclerosis, amyloidosis, an autoimmune disease, inflammation, a tumor, cancer, male impotence, wound healing, periodontal disease, neuropathy, retinopathy, nephropathy or neuronal degeneration (claimed).

Dwg.0/1

FS CPI

FA AB; DCN

MC CPI: B04-G01; B14-C03; B14-F06; B14-G02D; B14-H01; B14-J01A4; B14-N03; B14-N06B; B14-N10; **B14-N17B**; B14-S01; B14-S04; D05-H10; D05-H11

=> b home

FILE 'HOME' ENTERED AT 14:22:57 ON 21 OCT 2004

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=> d his

     (FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

     FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004
                E LONGLEY B/AU
L1            33 E4-6
                E LONGLEY J/AU
L2            12 E3,E9
L3           418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
                E SKIN DISEASE/CT
                E E4+ALL
                E E2
                E E3+ALL
L4         70942 "SKIN, DISEASE"+OLD,NT/CT
L5            11 L4 AND L1-2
L6            24 L4 AND L3
                E STEM CELL/CT
                E E3+ALL
L7         30857 STEM CELL+NT/CT
                E CELL/CT
                E E3+ALL
L8         11056 CELL+OLD,NT/CT (L) STEM
L9             6 L5-6 AND L7-8

     FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004
L10        48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC
                E STEM CELL#/BI
                E STEM CELL/BI
                E STEM CELL/ABEX
L11         4846 (STEM (1A) CELL?)/BIX
L12          710 L10 AND L11
                E LONGLEY B/AU
L13            3 E4
L14            1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
L15            1 L12 AND L13
L16          810 UYCO/PACO
L17            4 L12 AND (L14 OR L16)
L18            4 L15 OR L17

     FILE 'MEDLINE' ENTERED AT 14:28:32 ON 21 OCT 2004
L19       165235 STEM CELLS+NT/CT
L20       416171 SKIN DISEASES+NT/CT
L21        53892 L19/MAJ
L22       333514 L20/MAJ
L23       117556 L22 (L) (TH. OR PC)/CT
L24          113 L21 AND L23
                E LONGLEY B/AU
L25           47 E4-6
L26        49494 (COLUMBIA AND UNIV?)/CS
L27            0 L24 AND L25
L28            0 L24 AND L27
L29           70 L24 AND PY<=1999
                SEL AN 1-2 9-10 12-13 15-16 19 22 35-37
L30           13 E1-13 AND L29
L31            0 L24 AND L26

=>
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=> d his

     (FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

     FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004
                E LONGLEY B/AU
L1            33 E4-6
                E LONGLEY J/AU
L2            12 E3,E9
L3           418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
                E SKIN DISEASE/CT
                E E4+ALL
                E E2
                E E3+ALL
L4         70942 "SKIN, DISEASE"+OLD,NT/CT
L5            11 L4 AND L1-2
L6            24 L4 AND L3
                E STEM CELL/CT
                E E3+ALL
L7         30857 STEM CELL+NT/CT
                E CELL/CT
                E E3+ALL
L8         11056 CELL+OLD,NT/CT (L) STEM
L9             6 L5-6 AND L7-8

     FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004
L10        48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC
                E STEM CELL#/BI
                E STEM CELL/BI
                E STEM CELL/ABEX
L11         4846 (STEM (1A) CELL?)/BIX
L12          710 L10 AND L11
                E LONGLEY B/AU
L13            3 E4
L14            1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
L15            1 L12 AND L13
L16          810 UYCO/PACO
L17            4 L12 AND (L14 OR L16)
L18            4 L15 OR L17

     FILE 'MEDLINE' ENTERED AT 14:28:32 ON 21 OCT 2004
L19       165235 STEM CELLS+NT/CT
L20       416171 SKIN DISEASES+NT/CT
L21        53892 L19/MAJ
L22       333514 L20/MAJ
L23       117556 L22 (L) (TH. OR PC)/CT
L24          113 L21 AND L23
                E LONGLEY B/AU
L25           47 E4-6
L26        49494 (COLUMBIA AND UNIV?)/CS
L27            0 L24 AND L25
L28            0 L24 AND L27
L29           70 L24 AND PY<=1999
                SEL AN 1-2 9-10 12-13 15-16 19 22 35-37
L30           13 E1-13 AND L29

=> b medl
FILE 'MEDLINE' ENTERED AT 14:51:31 ON 21 OCT 2004

 FILE LAST UPDATED: 20 OCT 2004 (20041020/UP). FILE COVERS 1951 TO DATE.

 On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD
 for details. OLDMEDLINE now back to 1951.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2004 vocabulary. See http://www.nlm.nih.gov/mesh/ and
 http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a
 description of changes.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> d all l30 tot

L30  ANSWER 1 OF 13      MEDLINE on STN
AN   2001150874          MEDLINE
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DN PubMed ID: 11225638
TI Growth factors secreted by fibroblasts: role in healing diabetic foot ulcers.
AU Mansbridge J N; Liu K; Pinney R E; Patch R; Ratcliffe A; Naughton G K
CS Advanced Tissue Sciences, Inc., La Jolla, CA 92037, USA.. jonathan.mansbridge@advancedtissue.com
SO Diabetes, obesity & metabolism, **(1999 Sep)** 1 (5) 265-79. Ref: 109
Journal code: 100883645. ISSN: 1462-8902.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010315
CT Check Tags: Human
Animals
Cells, Cultured
Cytokines: SE, secretion
Diabetic Foot: PP, physiopathology
***Diabetic Foot: TH, therapy**
***Fibroblasts: PH, physiology**
Gene Expression
Growth Substances: GE, genetics
*Growth Substances: PH, physiology
Keratinocytes: PH, physiology
Macrophages: PH, physiology
Neovascularization, Physiologic
Skin: BS, blood supply
Skin Transplantation
Wound Healing
CN 0 (Cytokines); 0 (Growth Substances)

L30 ANSWER 2 OF 13 MEDLINE on STN
AN **2000427040** MEDLINE
DN PubMed ID: 10937098
TI Autologous fibroblasts for treatment of facial rhytids and dermal depressions. A pilot study.
AU Watson D; Keller G S; Lacombe V; Fodor P B; Rawnsley J; Lask G P
CS Division of Head and Neck Surgery, University of California, Los Angeles, USA.
SO Archives of facial plastic surgery : official publication for the American Academy of Facial Plastic and Reconstructive Surgery, Inc. and the International Federation of Facial Plastic Surgery Societies, **(1999 Jul-Sep)** 1 (3) 165-70.
Journal code: 100883500. ISSN: 1521-2491.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200009
ED Entered STN: 20000922
Last Updated on STN: 20000922
Entered Medline: 20000908
AB OBJECTIVE: To assess effectiveness of intradermal injections of autologous fibroblasts for the treatment of facial rhytids and dermal depressions. DESIGN: Six-month prospective pilot study. Photographs and silicone molds were taken of a prominent rhytid or dermal depression from each patient prior to treatment and at 6 months after treatment. SETTING: Specialty clinic in academic medical center. PATIENTS: Ten adults (age range, 24-69 years) who each exhibited a prominent rhytid or depressed facial scar. INTERVENTION: A 3-mm postauricular skin biopsy specimen from each participant was sent to Isolagen Technologies, Inc, laboratories, where a fibroblast cell line was developed. Three injection sessions were performed at 2-week intervals; target areas were the study site as well as behind the ear. MAIN OUTCOME MEASURES: Subjective improvement scores were obtained by each patient and 2 clinicians at every follow-up visit. Skin surface topographical features were evaluated with optical profilometry by comparing silicone molds before and after injection. Histological analysis was performed on a biopsy specimen of the postauricular injection site. RESULTS: Nine of 10 patients noted a 60% to 100% improvement with

the treatment; clinicians made similar observations. Size reduction of 10% up to 85% of the study site was demonstrated by optical profilometry for every patient. Microscopically, there was evidence of increased thickness and density of dermal-layer collagen. CONCLUSIONS: Intradermal injection of autologous fibroblasts may be an effective treatment option for facial rhytids and depressed scars.

CT Check Tags: Female; Human; Male
Acne Vulgaris: TH, therapy
Adult
Aged
***Cicatrix: TH, therapy**
Esthetics
***Facial Dermatoses: TH, therapy**
***Fibroblasts**
***Fibroblasts: TR, transplantation**
Follow-Up Studies
Injections, Intradermal
Middle Aged
Patient Satisfaction
Pilot Projects
Prospective Studies
Reconstructive Surgical Procedures
Transplantation, Autologous
Treatment Outcome

L30 ANSWER 3 OF 13 MEDLINE on STN
AN **1999116001** MEDLINE
DN PubMed ID: 9916172
TI Three-dimensional fibroblast culture implant for the treatment of diabetic foot ulcers: metabolic activity and therapeutic range.
AU Mansbridge J; Liu K; Patch R; Symons K; Pinney E
CS Advanced Tissue Sciences, Inc., La Jolla, California 92037, USA.
SO Tissue engineering, **(1998 Winter)** 4 (4) 403-14.
Journal code: 9505538. ISSN: 1076-3279.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990316
Last Updated on STN: 19990316
Entered Medline: 19990301
AB Dermagraft is three-dimensional, allogeneic, human neonatal dermal fibroblast culture grown on a degradable scaffold and cryopreserved. Clinical trials for treatment of diabetic foot ulcers showed optimal healing within a therapeutic range of metabolic activity, determined by 3[4,5-dimethylthiazol-2-yl]2,5-diphenyltetrazolium bromide (MTT) reduction. Actions of Dermagraft in wound repair include colonization by cells and provision of growth factors and cytokines, both activities dependent on living cells. Cells in the cryopreserved culture showed 60% viability by dye exclusion and, when isolated, were able to proliferate in monolayer culture. Protein synthesis by Dermagraft was inhibited 70-98% by cryopreservation, but, if within the therapeutic range, recovered to 45-85% of the prefreeze value over 48 h. Subtherapeutic Dermagraft showed variable, low recovery. Expression of mRNA for vascular endothelial growth factor (VEGF), platelet-derived growth factor A chain, and insulin-like growth factor-1 was reduced >83% in subtherapeutic compared with therapeutic Dermagraft. Granulocyte colony-stimulating factor and VEGF protein secretion, determined by enzyme-linked immunosorbent assay (ELISA), and angiogenic activity also depended on therapeutic range. VEGF secretion dropped sharply with MTT reductase in subtherapeutic tissue. The data demonstrate the critical dependence of the therapeutic properties of this living dermal implant on recovery of protein synthesis, growth factor expression, and angiogenesis, determined by metabolic activity.

CT Check Tags: Human; Male
Animals
Cell Survival
Cells, Cultured: TR, transplantation
Chick Embryo
Cryopreservation
Culture Media, Conditioned: CH, chemistry
***Diabetic Foot: SU, surgery**
Endothelial Growth Factors: BI, biosynthesis
Enzyme-Linked Immunosorbent Assay
Fibroblasts: ME, metabolism
***Fibroblasts: TR, transplantation**

Granulocyte Colony-Stimulating Factor: BI, biosynthesis
Infant, Newborn
Lymphokines: BI, biosynthesis
Neovascularization, Physiologic
RNA, Messenger: BI, biosynthesis
Skin, Artificial
Transplantation, Homologous
Vascular Endothelial Growth Factor A
Vascular Endothelial Growth Factors
Wound Healing
RN 143011-72-7 (Granulocyte Colony-Stimulating Factor)
CN 0 (Culture Media, Conditioned); 0 (Endothelial Growth Factors); 0 (Lymphokines); 0 (RNA, Messenger); 0 (Vascular Endothelial Growth Factor A); 0 (Vascular Endothelial Growth Factors)

L30 ANSWER 4 OF 13 MEDLINE on STN
AN **1999113022** MEDLINE
DN PubMed ID: 9893171
TI Studies on the biological activity of the dermal regeneration template.
AU Yannas I V
CS Departments of Mechanical Engineering and Materials Science and Engineering, Massachusetts Institute of Technology, Cambridge, Mass 02139-4307, USA.
SO Wound repair and regeneration : official publication of the Wound Healing Society [and] European Tissue Repair Society, **(1998 Nov-Dec)** 6 (6) 518-23. Ref: 42
Journal code: 9310939. ISSN: 1067-1927.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990222
AB In mammals, the skin normally responds to injury by the processes of wound contraction and scar formation. However, if a unique acellular dermal regeneration template is placed into the wound, wound contraction is strongly inhibited, and the skin undergoes a regenerative-type healing. It is proposed that these phenomena result from the disruption of a "mechanically coherent" organization of myofibroblasts within the wound. Evidence suggests that the dermal regeneration template prevents the formation of cell-extracellular matrix interactions (fibronexi), which foster the assemblage of a mechanically coherent wound bed. Additional studies support a similar mechanism for the enhanced regenerative-like response to peripheral nerves when extracellular matrix tubes are used to facilitate healing.
CT Check Tags: Human
Adult
Animals
Cicatrix: PP, physiopathology
***Cicatrix: PC, prevention & control**
Contracture: PP, physiopathology
*Contracture: PC, prevention & control
Extracellular Matrix: PH, physiology
***Fibroblasts: PH, physiology**
*Nerve Regeneration: PH, physiology
*Skin Physiology
Templates, Genetic
*Wound Healing: PH, physiology

L30 ANSWER 5 OF 13 MEDLINE on STN
AN **1998312640** MEDLINE
DN PubMed ID: 9650620
TI Corrective transduction of human epidermal stem cells in laminin-5-dependent junctional epidermolysis bullosa.
AU Dellambra E; Vailly J; Pellegrini G; Bondanza S; Golisano O; Macchia C; Zambruno G; Meneguzzi G; De Luca M
CS Laboratory of Tissue Engineering, I.D.I.-IRCCS, Istituto Dermopatico dell'Immacolata, Rome, Italy.
SO Human gene therapy, **(1998 Jun 10)** 9 (9) 1359-70.
Journal code: 9008950. ISSN: 1043-0342.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980925
Last Updated on STN: 19980925
Entered Medline: 19980916
AB Laminin-5 is composed of three distinct polypeptides, alpha3, beta3, and gamma2, which are encoded by three different genes, LAMA3, LAMB3, and LAMC2, respectively. We have isolated epidermal keratinocytes from a patient presenting with a lethal form of junctional epidermolysis bullosa characterized by a homozygous mutation of the LAMB3 gene, which led to complete absence of the beta3 polypeptide. In vitro, beta3-null keratinocytes were unable to synthesize laminin-5 and to assemble hemidesmosomes, maintained the impairment of their adhesive properties, and displayed a decrease of their colony-forming ability. A retroviral construct expressing a human beta3 cDNA was used to transduce primary beta3-null keratinocytes. Clonogenic beta3-null keratinocytes were transduced with an efficiency of 100%. Beta3-transduced keratinocytes were able to synthesize and secrete mature heterotrimeric laminin-5. Gene correction fully restored the keratinocyte adhesion machinery, including the capacity of proper hemidesmosomal assembly, and prevented the loss of the colony-forming ability, suggesting a direct link between adhesion to laminin-5 and keratinocyte proliferative capacity. Clonal analysis demonstrated that holoclones expressed the transgene permanently, suggesting stable correction of epidermal stem cells. Because cultured keratinocytes are used routinely to make autologous grafts for patients suffering from large skin or mucosal defects, the full phenotypic reversion of primary human epidermal stem cells defective for a structural protein opens new perspectives in the long-term treatment of genodermatoses.
CT Check Tags: Human; Support, Non-U.S. Gov't
Animals
Cells, Cultured
DNA: AN, analysis
Desmosomes: ME, metabolism
*Epidermis: CY, cytology
Epidermolysis Bullosa, Junctional: GE, genetics
Epidermolysis Bullosa, Junctional: PA, pathology
***Epidermolysis Bullosa, Junctional: TH, therapy**
Fluorescent Antibody Technique
*Gene Therapy
Genetic Vectors
Infant, Newborn
Keratinocytes: CY, cytology
Keratinocytes: UL, ultrastructure
Laminin: BI, biosynthesis
*Laminin: GE, genetics
Mice
Precipitin Tests
RNA: AN, analysis
Retroviridae: GE, genetics
***Stem Cells: CY, cytology**
*Transduction, Genetic
RN 63231-63-0 (RNA); 9007-49-2 (DNA)
CN 0 (Genetic Vectors); 0 (Laminin)

L30 ANSWER 6 OF 13 MEDLINE on STN
AN **1998271201** MEDLINE
DN PubMed ID: 9608274
TI [Prospects of use human fetal fibroblasts in the treatment of various etiology wounds].
Perspektivy ispol'zovaniia fetal'nykh fibroblastov cheloveka pri lechenii ran razlichnoi etiologii.
AU Kolokol'tseva T D; Iurchenko N D; Kolosov N G; Nechaeva E A; Shumakova O V; Khristo S A
SO Vestnik Rossiiskoi akademii meditsinskikh nauk / Rossiiskaia akademiia meditsinskikh nauk, **(1998)** (3) 32-5.
Journal code: 9215641. ISSN: 0869-6047.
CY RUSSIA: Russian Federation
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 199806
ED Entered STN: 19980708
Last Updated on STN: 19980708
Entered Medline: 19980622

AB The investigations have demonstrated a high efficiency of the certified human fetal lung fibroblasts in the treatment of skin wounds of various etiology. For treatment, a fibroblast monolayer grown on a backing was applied to various types of wounds (burns frostbites, donor site suppurations) and in shin phlegmon. There was wound debrediment and rapid skin recovery. Employing the cells from the certified cell culture banks provides not only a biologically and genetic uniform material, but also makes it possible to standardize treatments which are useful for wide medical application, by delivering the standard cells to medical institutions which have no experience in culturing. To set up banks of cell cultures and to store them for a long time at a temperature of liquid nitrogen in adequate quantities of ampoules allow the cultured fibroblasts to be provided with if a large quantity of cells is required, which may be useful in emergencies.

CT Check Tags: Human
Cell Division
Cell Transplantation
Cells, Cultured
English Abstract
*Fetal Tissue Transplantation
Fibroblasts: CY, cytology
***Fibroblasts: TR, transplantation**
Lung: CY, cytology
Lung: EM, embryology
***Skin Diseases: TH, therapy**
Treatment Outcome
Wound Healing
*Wounds and Injuries: TH, therapy

L30 ANSWER 7 OF 13 MEDLINE on STN
AN **1998180327** MEDLINE
DN PubMed ID: 9521032
TI New skin for old: developments in biological skin substitutes.
CM Comment on: Arch Dermatol. 1998 Mar;134(3):293-300. PubMed ID: 9521027
AU Phillips T J
SO Archives of dermatology, **(1998 Mar)** 134 (3) 344-9.
Journal code: 0372433. ISSN: 0003-987X.
CY United States
DT Commentary
Editorial
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199804
ED Entered STN: 19980416
Last Updated on STN: 19980416
Entered Medline: 19980409
CT Check Tags: Human
Cells, Cultured
***Fibroblasts: TR, transplantation**
Graft Rejection
*Keratinocytes: TR, transplantation
Risk Factors
Skin Transplantation
*Skin, Artificial
Skin, Artificial: AE, adverse effects
***Varicose Ulcer: TH, therapy**

L30 ANSWER 8 OF 13 MEDLINE on STN
AN **1998180321** MEDLINE
DN PubMed ID: 9521027
TI Rapid healing of venous ulcers and lack of clinical rejection with an allogeneic cultured human skin equivalent. Human Skin Equivalent Investigators Group.
CM Comment in: Arch Dermatol. 1998 Mar;134(3):344-9. PubMed ID: 9521032
Comment in: Arch Dermatol. 1998 Nov;134(11):1483-4. PubMed ID: 9828891
AU Falanga V; Margolis D; Alvarez O; Auletta M; Maggiacomo F; Altman M; Jensen J; Sabolinski M; Hardin-Young J
CS Department of Dermatology and Cutaneous Surgery, University of Miami School of Medicine, Fla 33136, USA.
SO Archives of dermatology, **(1998 Mar)** 134 (3) 293-300.
Journal code: 0372433. ISSN: 0003-987X.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)

LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199804
ED Entered STN: 19980416
Last Updated on STN: 20000303
Entered Medline: 19980409
AB OBJECTIVE: To test the safety, efficacy, and immunological impact of a cultured allogeneic human skin equivalent (HSE) in the treatment of venous ulcers. DESIGN: Prospective, randomized study. SETTING: Multicenter study in the outpatient setting. INTERVENTION: Each patient with a venous ulcer received either compression therapy alone or compression therapy and treatment with HSE. The patients were evaluated for HSE safety, complete (100%) ulcer healing, time to wound closure, wound recurrence, and immune response to the HSE. OUTCOME: The study was completed as planned in 293 randomized patients. RESULTS: Treatment with HSE was more effective than compression therapy in the percentage of patients healed by 6 months (63% vs 49%; P=.02, Fisher exact test, 2-tailed) and the median time to complete wound closure (61 days vs 181 days; P=.003, log-rank test). Treatment with HSE was superior to compression therapy in healing larger (> 1000 mm2; P=.02) and deeper ulcers (P=.003) and ulcers of more than 6 months' duration (P=.001). Occurrence of adverse events was similar in both groups. No symptoms or signs of rejection occurred in response to treatment with HSE, and no HSE-specific immune responses were detected in vitro to bovine collagen or to alloantigens expressed on keratinocytes or fibroblasts. CONCLUSIONS: Treatment with HSE healed venous ulcers more rapidly and in more patients than compression therapy alone. There was no clinical or laboratory evidence of rejection or sensitization in response to HSE application. These data suggest that HSE represents a significant advance in the treatment of venous ulcers, particularly those that are difficult to heal.
CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't
Aged
Animals
Antibodies: AN, analysis
Bandages
Cattle
Collagen: IM, immunology
Cytotoxicity, Immunologic
***Fibroblasts: TR, transplantation**
Graft Rejection
HLA Antigens: IM, immunology
*Keratinocytes: TR, transplantation
Prospective Studies
Recurrence
*Skin, Artificial
***Varicose Ulcer: TH, therapy**
Wound Healing
RN 9007-34-5 (Collagen)
CN 0 (Antibodies); 0 (HLA Antigens)

L30 ANSWER 9 OF 13 MEDLINE on STN
AN **97420209** MEDLINE
DN PubMed ID: 9274713
TI The importance of epidermal stem cells in keratinocyte-mediated gene therapy.
AU De Luca M; Pellegrini G
SO Gene therapy, **(1997 May)** 4 (5) 381-3.
Journal code: 9421525. ISSN: 0969-7128.
CY ENGLAND: United Kingdom
DT Editorial
LA English
FS Priority Journals
EM 199709
ED Entered STN: 19971008
Last Updated on STN: 19971008
Entered Medline: 19970919
CT Check Tags: Human
*Burns: TH, therapy
Clone Cells
*Epidermis: CY, cytology
***Epidermolysis Bullosa: TH, therapy**
*Gene Therapy: MT, methods
Interleukin-6: GE, genetics
*Keratinocytes
Skin Transplantation
***Stem Cells**

Transduction, Genetic
CN 0 (Interleukin-6)

L30 ANSWER 10 OF 13 MEDLINE on STN
AN **97182109** MEDLINE
DN PubMed ID: 9030156
TI Single exposures to 5-fluorouracil: a possible mode of targeted therapy to reduce contractile scarring in the injured tendon.
AU Khan U; Occleston N L; Khaw P T; McGrouther D A
CS Department of Plastic and Reconstructive Surgery, University College, London, England.
SO Plastic and reconstructive surgery, **(1997 Feb)** 99 (2) 465-71.
Journal code: 1306050. ISSN: 0032-1052.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199703
ED Entered STN: 19970407
Last Updated on STN: 19970407
Entered Medline: 19970324
AB After injury, adhesions may develop between the digital flexor tendons and their sheaths. Fibroblasts are key cells in this fibrotic adhesive process, and two possible sources for these cells are the synovial sheath and the endotenon tissue (tendon core). Fibroblasts seeded into a collagen lattice will contract the collagen. This fibroblast-populated collagen lattice contraction was used to investigate the ability of the fibroblasts from the synovial sheath and endotenon to reorganize collagen (an important function in the formation of adhesions). Endotenon and synovial fibroblasts isolated from 30 animals were used in the study. Synovial fibroblasts produced significantly greater collagen lattice contraction compared with endotenon fibroblasts ($p < 0.05$). The possibility of preventing collagen lattice contraction with a single, nontoxic 5-minute treatment of the fibroblast-populated collagen lattices with the antimetabolite 5-fluorouracil was investigated. Compared with controls the degree of fibroblast-populated collagen lattice contraction was significantly inhibited ($p < 0.05$) with the use of 5-fluorouracil for endotenon and synovial cells. These results demonstrate the potential for locally targeted therapy in tendon healing. Because of the different contractile properties of the two cell lines, a change in the balance between intrinsic and extrinsic healing might be achieved with this method of therapy; in turn, this might lead to better functional results following surgery.
CT Check Tags: Support, Non-U.S. Gov't
Animals
*Antimetabolites: AD, administration & dosage
Cells, Cultured
Cicatrix: ET, etiology
***Cicatrix: PC, prevention & control**
*Collagen: DE, drug effects
*Collagen: PH, physiology
Cytoskeleton: DE, drug effects
***Fibroblasts: DE, drug effects**
Fibroblasts: PH, physiology
*Fluorouracil: AD, administration & dosage
Rabbits
*Tendon Injuries: CO, complications
Tendon Injuries: PA, pathology
RN 51-21-8 (Fluorouracil); 9007-34-5 (Collagen)
CN 0 (Antimetabolites)

L30 ANSWER 11 OF 13 MEDLINE on STN
AN **94171837** MEDLINE
DN PubMed ID: 8126023
TI Effects of hyaluronan on collagen fibrillar matrix contraction by fibroblasts.
AU Huang-Lee L L; Wu J H; Nimni M E
CS Department of Biochemistry, School of Medicine, University of Southern California, Los Angeles.
SO Journal of biomedical materials research, **(1994 Jan)** 28 (1) 123-32.
Journal code: 0112726. ISSN: 0021-9304.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 199404
ED Entered STN: 19940420
Last Updated on STN: 19940420
Entered Medline: 19940412
AB Hyaluronan, found in high concentrations in fetal tissues, appears to have a major role in preventing scar formation in fetal wounds. Nevertheless, its role in inhibiting wound contractures associated with scar formation has not been clearly demonstrated. Our current study evaluated the effects of hyaluronan using an in vitro floating collagen fibrillar matrix (CFM) contraction model. The results demonstrated that the contraction of CFM by fibroblasts was significantly reduced when high concentrations (> 1 mg/mL) of hyaluronan were present in the media. This phenomenon is unique to hyaluronan, because chondroitin sulfate was ineffective in this connection. Fibroblast migration and proliferation studies indicated that high concentrations of hyaluronan stimulated cell migration and had no cytotoxic effects. Some possible mechanisms by which high concentrations of hyaluronan reduced CFM contraction by fibroblasts were proposed. Because the viscosity of a hyaluronan solution is much greater than that of chondroitin sulfate, and this increases with concentration, we investigated whether this property in itself was an important factor in inhibiting CFM contraction. No direct correlation was found between the viscosity of glycosaminoglycans and their ability to reduce CFM contraction.
CT Check Tags: Human
Animals
Cattle
Cell Division: DE, drug effects
Cell Movement: PH, physiology
Cells, Cultured
Chondroitin Sulfates: PD, pharmacology
***Cicatrix: PC, prevention & control**
*Collagen: DE, drug effects
DNA: ME, metabolism
***Fibroblasts: DE, drug effects**
Fibroblasts: UL, ultrastructure
Glycosaminoglycans: CH, chemistry
Glycosaminoglycans: IP, isolation & purification
*Hyaluronic Acid: PD, pharmacology
Viscosity
RN 9004-61-9 (Hyaluronic Acid); 9007-28-7 (Chondroitin Sulfates); 9007-34-5 (Collagen); 9007-49-2 (DNA)
CN 0 (Glycosaminoglycans)

L30 ANSWER 12 OF 13 MEDLINE on STN
AN **94096931** MEDLINE
DN PubMed ID: 8271751
TI [Treatment of chronic non-healing donor sites: transplantation of cultured human fibroblasts].
Lechenie dlitelno ne zazhivaiushchikh donorskikh uchastkov: transplan tatsiia kultivirovannykh allofibroblastov cheloveka.
AU Moroz V Iu; Grishkevich V M; Alekseev A A; Glushchenko E V; Sarkisov D S; Gurukov Sh R; Tumanov V P; Morozov S S
SO Khirurgiia, **(1993 Jul)** (7) 71-5.
Journal code: 0412765. ISSN: 0023-1207.
CY RUSSIA: Russian Federation
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 199402
ED Entered STN: 19940215
Last Updated on STN: 19940215
Entered Medline: 19940203
AB The authors discuss the first results of the use of cultivated human allofibroblasts in the treatment of persisting wounds of the donor areas in 13 patients with deep burns and cicatricial deformities caused by the burns. Transplantation of cell cultures led to complete epithelialization of the wounds in 6-10 days. Complication, suppuration of the grafted material, was recorded in one case. Reaction of graft rejection was not encountered. The authors believe that the effect of the transplantation is due to stimulation of the epithelialization processes by the cultivated fibroblasts.
CT Check Tags: Human
Burns: CO, complications
*Burns: SU, surgery
Cells, Cultured
Chronic Disease

Cicatrix: ET, etiology
Cicatrix: PP, physiopathology
***Cicatrix: SU, surgery**
English Abstract
***Fibroblasts: TR, transplantation**
Reoperation
*Skin Transplantation
Treatment Outcome
Wound Healing

L30 ANSWER 13 OF 13 MEDLINE on STN
AN **94058308** MEDLINE
DN PubMed ID: 8239704
TI Keratinocyte gene therapy.
AU Vogel J C
CS Dermatology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Md.
SO Archives of dermatology, **(1993 Nov)** 129 (11) 1478-83. Ref: 52
Journal code: 0372433. ISSN: 0003-987X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931214
AB BACKGROUND: Gene therapy is currently being used in clinical trials to treat a variety of diseases. In keratinocyte gene therapy, the gene that will correct the disease by expressing the normal protein or enzyme is inserted and expressed in keratinocytes. Keratinocytes have significant potential, as a target cell of gene therapy, in the treatment of both systemic diseases as well as skin diseases caused by a genetic defect in keratinocytes. OBSERVATIONS: Although keratinocyte gene therapy is not yet being tested in clinical trials, animal models do exist where keratinocytes are being used to secrete factors such as human growth hormone and factor IX (for hemophilia) into the systemic circulation. Genetic diseases of the skin such as recessive epidermolysis bullosa dystrophica or xeroderma pigmentosum have not yet been treated with keratinocyte gene therapy in animal models. CONCLUSIONS: Keratinocytes have many advantages as a target cell in gene therapy, and progress has been made using animal models. However, the sustained and efficient delivery of factors to the bloodstream by keratinocytes expressing a transgene has not yet been accomplished. Future goals are to obtain adequate levels of the desired factors, hormones, or enzymes for sustained periods of time, either in keratinocytes or in the vascular system.
CT Check Tags: Human
Animals
Basement Membrane: ME, metabolism
Factor IX: ME, metabolism
***Fibroblasts**
Forecasting
Gene Expression
*Gene Therapy: MT, methods
Genetic Vectors
Growth Hormone: BL, blood
*Keratinocytes
Models, Biological
Mutation
Skin Diseases: BL, blood
Skin Diseases: GE, genetics
***Skin Diseases: TH, therapy**
Tissue Culture
RN 9001-28-9 (Factor IX); 9002-72-6 (Growth Hormone)
CN 0 (Genetic Vectors)

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